

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options:

VISIT...

LANZAROTE
Caliente.COM

	ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- ≤ 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent = high dose.

Asthma: acute severe

Patients with acute severe asthma are stratified into moderate, severe or life-threatening

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

British Thoracic Society guidelines

- magnesium sulphate recommended as next step for patients who are not responding (e.g. 1.2 - 2g IV over 20 mins)
- little evidence to support use of IV aminophylline (although still mentioned in management plans)
- if no response consider IV salbutamol

Asthma: diagnosis

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

NICE guidance has radically changed how asthma should be diagnosed. It advocates moving away from subjective/clinical judgements are more towards **objective** tests.

There is particular emphasis on the use of **fractional exhaled nitric oxide (FeNO)**. Nitric oxide is produced by 3 types of nitric oxide synthases (NOS). One of the types is inducible (iNOS) and levels tend to rise in inflammatory cells, particularly eosinophils. Levels of NO therefore typically correlate with levels of inflammation.

Other more established objective tests such as spirometry and peak flow variability are still important.

All patients ≥ 5 years should have objective tests. Once a child with suspected asthma reaches the age of 5 years objective tests should be performed to confirm the diagnosis.

Diagnostic testing

Diagnostic testing

Patients ≥ 17 years

- patients should be asked if their symptoms are better on days away from work/during holidays. If so, patients should be referred to a specialist as possible occupational asthma
- all patients should have spirometry with a bronchodilator reversibility (BDR) test
- all patients should have a FeNO test

Patients 5-16 years

- all patients should have spirometry with a bronchodilator reversibility (BDR) test
- a FeNO test should be requested if there is normal spirometry or obstructive spirometry with a negative bronchodilator reversibility (BDR) test

Patients < 5 years

- diagnosis should be made on clinical judgement

Specific points about the tests

FeNO

- in adults level of ≥ 40 parts per billion (ppb) is considered positive
- in children a level of ≥ 35 parts per billion (ppb) is considered positive

Spirometry

- FEV1/FVC ratio less than 70% (or below the lower limit of normal if this value is available) is considered obstructive

Reversibility testing

- in adults, a positive test is indicated by an improvement in FEV1 of 12% or more and increase in volume of 200 ml or more
 - in children, a positive test is indicated by an improvement in FEV1 of 12% or more
-

Asthma: occupational

Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.

Exposure to the following chemicals is associated with occupational asthma:

- isocyanates - the most common cause. Example occupations include spray painting and foam moulding using adhesives
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

Serial measurements of peak expiratory flow are recommended at work and away from work.

Referral should be made to a respiratory specialist for patients with suspected occupational asthma.

Leukotriene receptor antagonists

Basics

- e.g. Montelukast, zafirlukast
- have both anti-inflammatory and bronchodilatory properties
- should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting β_2 -agonist
- particularly useful in aspirin-induced asthma
- associated with the development of Churg-Strauss syndrome

Next question >

Pulmonary arterial hypertension: causes and classification

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise. PAH has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)

- idiopathic*
- familial
- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, HIV**, drugs and toxins, sickle cell disease
- persistent pulmonary hypertension of the newborn

Group 2: Pulmonary hypertension with left heart disease

- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia

- COPD
- interstitial lung disease
- sleep apnoea
- high altitude

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions

- lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

*previously termed primary pulmonary hypertension

**the mechanism by which HIV infection produces pulmonary hypertension remains unknown



ARDS

Acute respiratory distress syndrome (ARDS) is caused by the increased permeability of alveolar capillaries leading to fluid accumulation in the alveoli, i.e. non-cardiogenic pulmonary oedema. It is a serious condition that has a mortality of around 40% and is associated with significant morbidity in those who survive.

Causes

- infection: sepsis, pneumonia
- massive blood transfusion
- trauma
- smoke inhalation
- pancreatitis
- cardio-pulmonary bypass

Clinical features are typically of an acute onset and severe:

- dyspnoea
- elevated respiratory rate
- bilateral lung crackles
- low oxygen saturations

A chest x-ray and arterial blood gases are the key investigations.

Criteria (American-European Consensus Conference)

- acute onset (within 1 week of a known risk factor)
- pulmonary oedema: bilateral infiltrates on chest x-ray ('not fully explained by effusions, lobar/lung collapse or nodules)
- non-cardiogenic (pulmonary artery wedge pressure needed if doubt)
- $pO_2/FiO_2 < 40kPa$ (200 mmHg)

Management

- due to the severity of the condition patients are generally managed in ITU
- oxygenation/ventilation to treat the hypoxaemia
- general organ support e.g. vasopressors as needed
- treatment of the underlying cause e.g. antibiotics for sepsis
- certain strategies such as prone positioning and muscle relaxation have been shown to improve outcome in ARDS

Tuberculosis

Tuberculosis (TB) is an infection caused by *Mycobacterium tuberculosis* that most commonly affects the lungs. Understanding the pathophysiology of TB can be difficult - the key is to differentiate between primary and secondary disease.

Primary tuberculosis

A non-immune host who is exposed to *M. tuberculosis* may develop primary infection of the lungs. A small lung lesion known as a Ghon focus develops. The Ghon focus is composed of tubercle-laden macrophages. The combination of a Ghon focus and hilar lymph nodes is known as a Ghon complex

In immunocompetent people the initially lesion usually heals by fibrosis. Those who are immunocompromised may develop disseminated disease (miliary tuberculosis).

Secondary (post-primary) tuberculosis

If the host becomes immunocompromised the initial infection may become reactivated. Reactivation generally occurs in the apex of the lungs and may spread locally or to more distant sites. Possible causes of immunocompromise include:

- immunosuppressive drugs including steroids
- HIV
- malnutrition

The lungs remain the most common site for secondary tuberculosis. Extra-pulmonary infection may occur in the following areas:

- central nervous system (tuberculous meningitis - the most serious complication)
- vertebral bodies (Pott's disease)
- cervical lymph nodes (scrofuloderma)
- renal
- gastrointestinal tract



Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a $10 * 5 = 50$ times increased risk

Lung cancer: small cell

Features

- usually central
- arise from APUD* cells
- associated with ectopic ADH, ACTH secretion
- ADH → hyponatraemia
- ACTH → Cushing's syndrome
- ACTH secretion can cause bilateral adrenal hyperplasia, the high levels of cortisol can lead to hypokalaemic alkalosis
- Lambert-Eaton syndrome: antibodies to voltage gated calcium channels causing myasthenic like syndrome

Management

- usually metastatic disease by time of diagnosis
- patients with very early stage disease (T1-2a, N0, M0) are now considered for surgery. NICE support this approach in their 2011 guidelines
- however, most patients with limited disease receive a combination of chemotherapy and radiotherapy
- patients with more extensive disease are offered palliative chemotherapy



Lung cancer: non-small cell management

Management

- only 20% suitable for surgery
- mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- curative or palliative radiotherapy
- poor response to chemotherapy

Surgery contraindications

- assess general health
- stage IIIb or IV (i.e. metastases present)
- FEV1 < 1.5 litres is considered a general cut-off point*
- malignant pleural effusion
- tumour near hilum
- vocal cord paralysis
- SVC obstruction

* However if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results

Lung cancer: investigation

Chest x-ray

- this is often the first investigation done in patients with suspected lung cancer
- in around 10% of patients subsequently diagnosed with lung cancer the chest x-ray was reported as normal

CT

- is the investigation of choice to investigate suspected lung cancer

Bronchoscopy

- this allows a biopsy to be taken to obtain a histological diagnosis sometimes aided by endobronchial ultrasound

PET scanning

- is typically done in non-small cell lung cancer to establish eligibility for curative treatment
- uses 18-fluorodeoxyglucose which is preferentially taken up by neoplastic tissue
- has been shown to improve diagnostic sensitivity of both local and distant metastasis spread in non-small cell lung cancer

Next question >

Lung cancer: referral

The 2015 NICE cancer referral guidelines gave the following advice:

Refer people using a suspected cancer pathway referral (for an appointment within 2 weeks) for lung cancer if they:

- have chest x-ray findings that suggest lung cancer
- are aged 40 and over with unexplained haemoptysis

Offer an urgent chest x-ray (to be performed within 2 weeks) to assess for lung cancer in people aged 40 and over if they have 2 or more of the following unexplained symptoms, or if they have ever smoked and have 1 or more of the following unexplained symptoms:

- cough
- fatigue
- shortness of breath
- chest pain
- weight loss
- appetite loss

Consider an urgent chest x-ray (to be performed within 2 weeks) to assess for lung cancer in people aged 40 and over with any of the following:

- persistent or recurrent chest infection
- finger clubbing
- supraclavicular lymphadenopathy or persistent cervical lymphadenopathy
- chest signs consistent with lung cancer
- thrombocytosis

Next question >

Lung cancer: carcinoid

The vast majority of bronchial adenomas are carcinoid tumours, arising from the amine precursor uptake and decarboxylation (APUD) system, like small cell tumours. Lung carcinoid accounts 1% of lung tumours and for 10% of carcinoid tumours. The term bronchial adenoma is being phased out.

Lung carcinoid

- typical age = 40-50 years
- smoking not risk factor
- slow growing: e.g. long history of cough, recurrent haemoptysis
- often centrally located and not seen on CXR
- 'cherry red ball' often seen on bronchoscopy
- carcinoid syndrome itself is rare (associated with liver metastases)

Management

- surgical resection
 - if no metastases then 90% survival at 5 years
- 

Lung cancer: paraneoplastic features

Small cell

- ADH
- ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome

Squamous cell

- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)
- hyperthyroidism due to ectopic TSH

Adenocarcinoma

- gynaecomastia
- hypertrophic pulmonary osteoarthropathy (HPOA)



Hypertrophic pulmonary osteoarthropathy is a proliferative periostitis involving that typically involves the long bones. It is often painful.

*whilst it is traditionally taught that HPOA is most common with squamous cell carcinoma some studies indicate that adenocarcinoma is the most common cause e.g. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4049689/>

Mesothelioma

Features

- Dyspnoea, weight loss, chest wall pain
- Clubbing
- 30% present as painless pleural effusion
- Only 20% have pre-existing asbestosis
- History of asbestos exposure in 85-90%, latent period of 30-40 years

Basics

- Malignancy of mesothelial cells of pleura
- Metastases to contralateral lung and peritoneum
- Right lung affected more often than left

Investigation/diagnosis

- suspicion is normally raised by a chest x-ray showing either a pleural effusion or pleural thickening
- the next step is normally a pleural CT
- if a pleural effusion is present fluid should be sent for MC&S, biochemistry and cytology (but cytology is only helpful in 20-30% of cases)
- local anaesthetic thoracoscopy is increasingly used to investigate cytology negative exudative effusions as it has a high diagnostic yield (around 95%)
- if an area of pleural nodularity is seen on CT then an image-guided pleural biopsy may be used

Management

- Symptomatic
- Industrial compensation
- Chemotherapy, Surgery if operable
- Prognosis poor, median survival 12 months

Next question >

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams
- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline
- contraindicated in pregnancy and breast feeding

Bupropion

- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because '*some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.*'. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions

- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Microscopic polyangiitis

Microscopic polyangiitis is a small-vessel ANCA vasculitis

Features

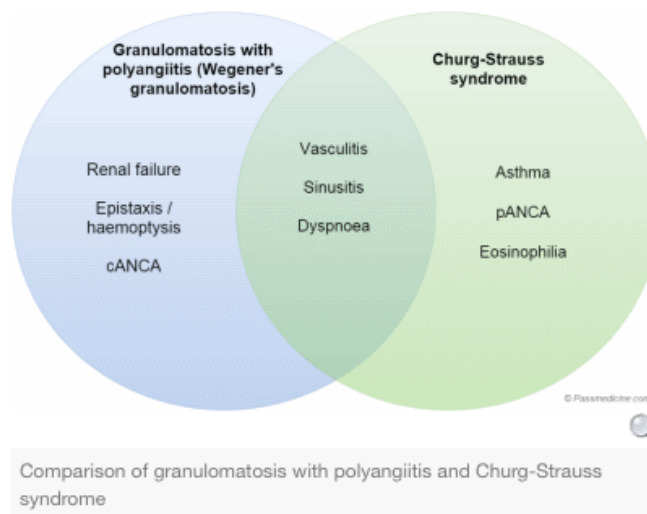
- renal impairment: raised creatinine, haematuria, proteinuria
 - fever
 - other systemic symptoms: lethargy, myalgia, weight loss
 - rash: palpable purpura
 - cough, dyspnoea, haemoptysis
 - mononeuritis multiplex
-

Granulomatosis with polyangiitis (Wegener's granulomatosis)

Granulomatosis with polyangiitis is now the preferred term for Wegener's granulomatosis. It is an autoimmune condition associated with a necrotizing granulomatous vasculitis, affecting both the upper and lower respiratory tract as well as the kidneys.

Features

- upper respiratory tract: epistaxis, sinusitis, nasal crusting
- lower respiratory tract: dyspnoea, haemoptysis
- rapidly progressive glomerulonephritis ('pauci-immune', 80% of patients)
- saddle-shape nose deformity
- also: vasculitic rash, eye involvement (e.g. proptosis), cranial nerve lesions



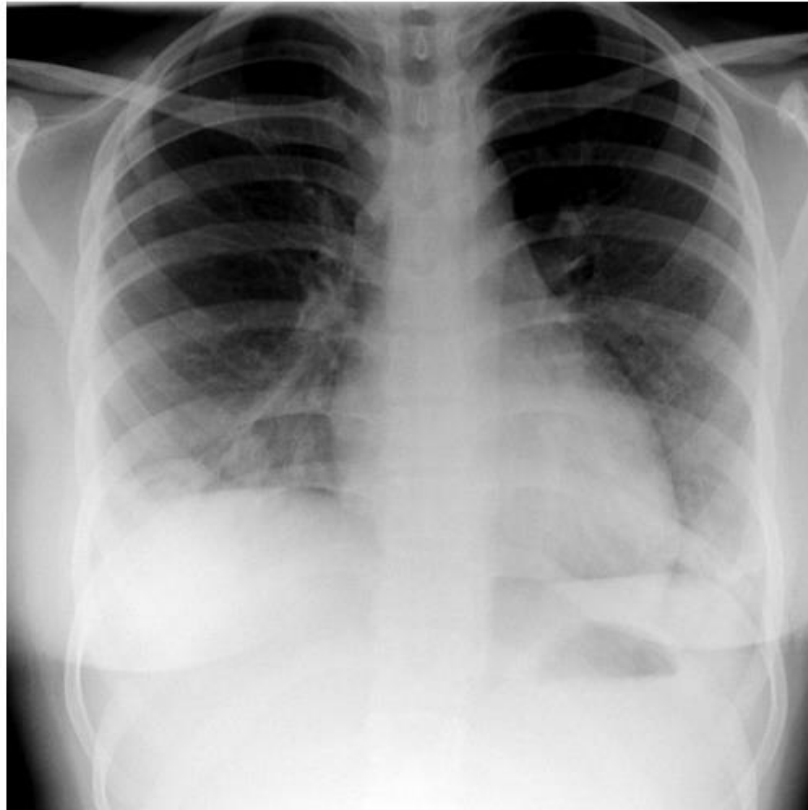
Investigations

- cANCA positive in > 90%, pANCA positive in 25%
- chest x-ray: wide variety of presentations, including cavitating lesions
- renal biopsy: epithelial crescents in Bowman's capsule

Management

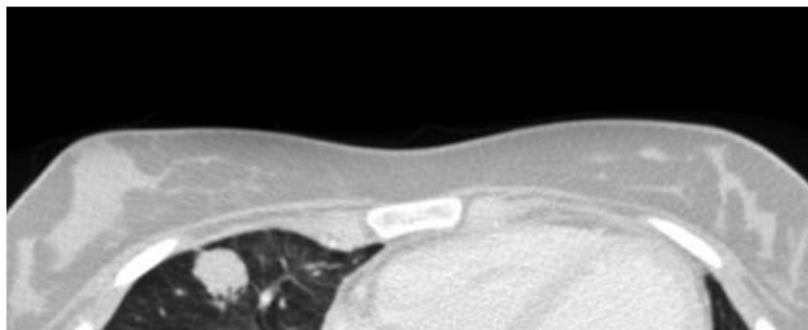
- steroids
- cyclophosphamide (90% response)
- plasma exchange
- median survival = 8-9 years

- plasma exchange
- median survival = 8-9 years



© Image used on license from Radiopaedia

Chest x-ray from a young male patient with granulomatosis with polyangiitis. Whilst the changes are subtle it demonstrates a number of ill-defined nodules the largest of which projects over the dome of the right hemidiaphragm. This nodule appears to have a central lucency suggesting cavitation





© Image used on license from Radiopaedia

Chest x-ray from a young male patient with granulomatosis with polyangiitis. Whilst the changes are subtle it demonstrates a number of ill-defined nodules the largest of which projects over the dome of the right hemidiaphragm. This nodule appears to have a central lucency suggesting cavitation



© Image used on license from Radiopaedia

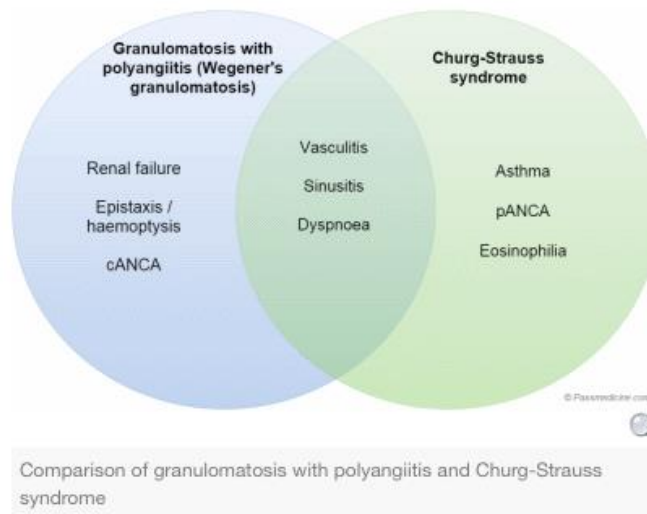
CT of the same patient showing the changes in a much more obvious way, confirming the presence of at least 2 nodules, the larger of the two having a large central cavity and and air-fluid level

Churg-Strauss syndrome

Churg-Strauss syndrome is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Leukotriene receptor antagonists may precipitate the disease

Pulmonary eosinophilia

Causes of pulmonary eosinophilia

- Churg-Strauss syndrome
- allergic bronchopulmonary aspergillosis (ABPA)
- Löffler's syndrome
- eosinophilic pneumonia
- hypereosinophilic syndrome
- tropical pulmonary eosinophilia
- drugs: nitrofurantoin, sulphonamides
- less common: Wegener's granulomatosis

Löffler's syndrome

- transient CXR shadowing and blood eosinophilia
- thought to be due to parasites such as *Ascaris lumbricoides* causing an alveolar reaction
- presents with a fever, cough and night sweats which often last for less than 2 weeks.
- generally a self-limiting disease

Tropical pulmonary eosinophilia

- associated with *Wuchereria bancrofti* infection



Oxygen dissociation curve

The oxygen dissociation curve describes the relationship between the percentage of saturated haemoglobin and partial pressure of oxygen in the blood. It is not affected by haemoglobin concentration

Basics

- shifts to left = for given oxygen tension there is increased saturation of Hb with oxygen i.e. decreased oxygen delivery to tissues
- shifts to right = for given oxygen tension there is reduced saturation of Hb with oxygen i.e. enhanced oxygen delivery to tissues

Shifts to Left = Lower oxygen delivery	Shifts to Right = Raised oxygen delivery
HbF, methaemoglobin, carboxyhaemoglobin Low [H ⁺] (alkali) Low pCO ₂ Low 2,3-DPG Low temperature	Raised [H ⁺] (acidic) Raised pCO ₂ Raised 2,3-DPG* Raised temperature

The L rule

Shifts to **L** → Lower oxygen delivery, caused by

- Low [H⁺] (alkali)
- Low pCO₂
- Low 2,3-DPG
- Low temperature

Another mnemonic is '**CADET**, face Right!' for **C**O₂, **A**cid, 2,3-**D**PG, **E**xercise and **T**emperature

*2,3-diphosphoglycerate



Transfer factor



The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO

- asthma
- pulmonary haemorrhage (Wegener's, Goodpasture's)
- left-to-right cardiac shunts
- polycythaemia
- hyperkinetic states
- male gender, exercise

Causes of a lower TLCO

- pulmonary fibrosis
- pneumonia
- pulmonary emboli
- pulmonary oedema
- emphysema
- anaemia
- low cardiac output

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

B*I*U**A**

T!

Save my notes

ATS Concise Clinical Review

4 1

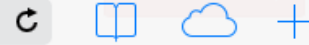
Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

Pulmonary function tests

Pulmonary function tests can be used to determine whether a respiratory disease is obstructive or restrictive. The table below summarises the main findings and gives some example conditions:

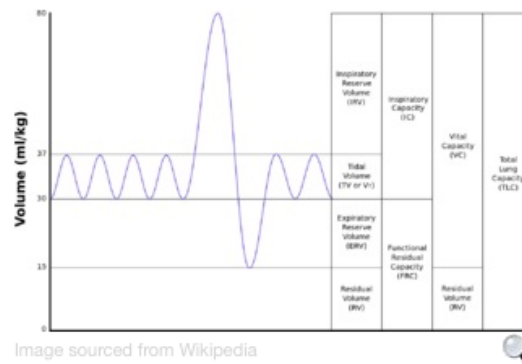
Obstructive lung disease	Restrictive lung disease
FEV1 - significantly reduced FVC - reduced or normal FEV1% (FEV1/FVC) - reduced	FEV1 - reduced FVC - significantly reduced FEV1% (FEV1/FVC) - normal or increased
Asthma COPD Bronchiectasis Bronchiolitis obliterans	Pulmonary fibrosis Asbestosis Sarcoidosis Acute respiratory distress syndrome Infant respiratory distress syndrome Kyphoscoliosis Neuromuscular disorders

Next question >



Respiratory physiology: lung volumes

The diagram below demonstrates the lung volumes which may be measured:



Tidal volume (TV)

- volume inspired or expired with each breath at rest
- 500ml in males, 350ml in females

Inspiratory reserve volume (IRV) = 2-3 L

- maximum volume of air that can be inspired at the end of a normal tidal inspiration
- inspiratory capacity = TV + IRV

Expiratory reserve volume (ERV) = 750ml

- maximum volume of air that can be expired at the end of a normal tidal expiration

Residual volume (RV) = 1.2L

- volume of air remaining after maximal expiration
- increases with age
- $RV = FRC - ERV$

Vital capacity (VC) = 5L

- maximum volume of air that can be expired after a maximal inspiration
- 4,500ml in males, 3,500 mls in females
- decreases with age
- $VC = \text{inspiratory capacity} + ERV$

Total lung capacity (TLC) is the sum of the vital capacity + residual volume

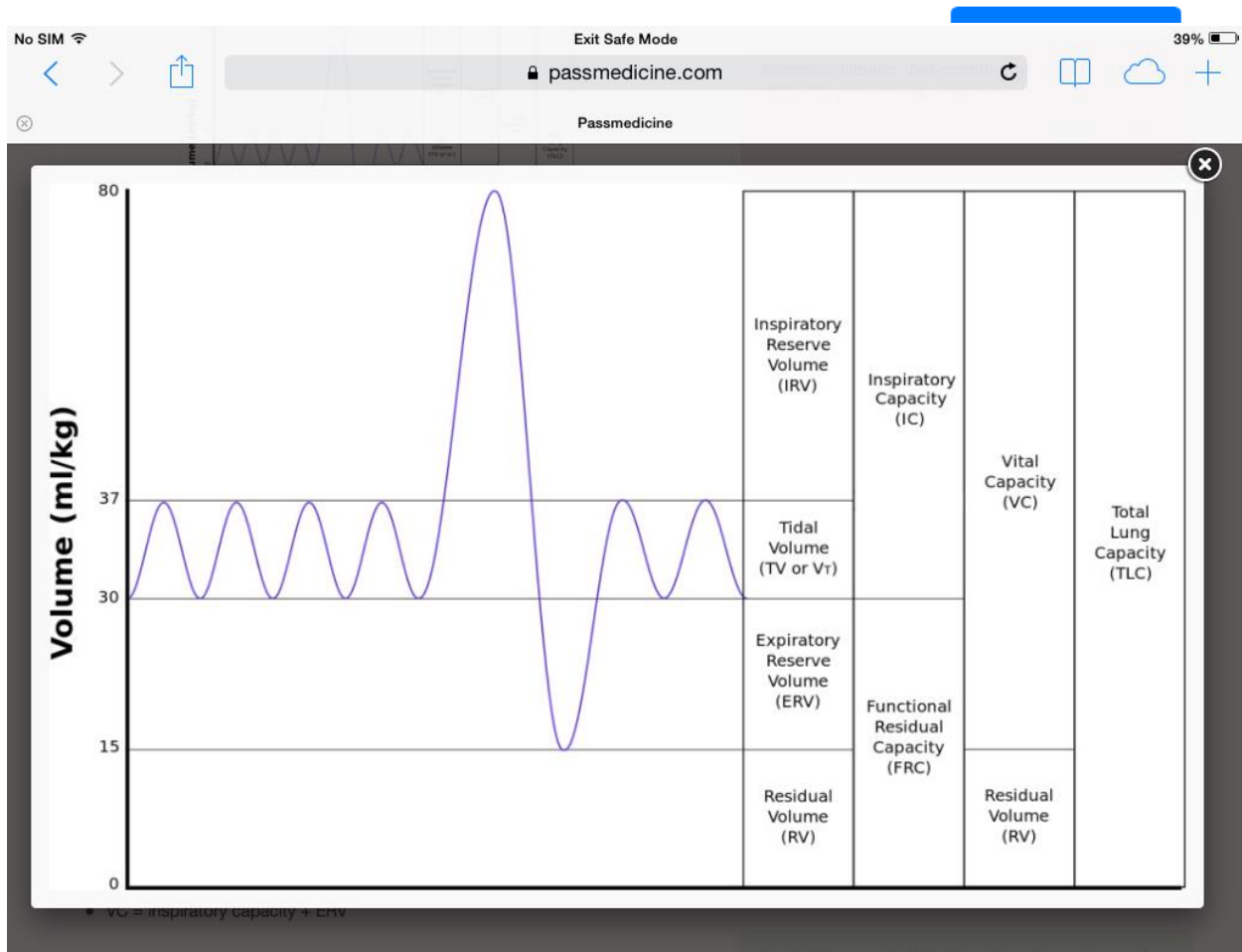
Physiological dead space (V_D)

- $V_D = \text{tidal volume} \times (\text{PaCO}_2 - \text{PeCO}_2) / \text{PaCO}_2$
- where PeCO_2 = expired air CO_2

Flow volume loop

A normal flow volume loop is often described as a 'triangle on top of a semi circle'

Flow volume loops are the most suitable way of assessing compression of the upper airway



Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35*
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H₂O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H₂O whilst BTS suggest 12-15 cm H₂O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

*the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used

Next question >

Respiratory acidosis

Respiratory acidosis may be caused by a number of conditions

- COPD
- decompensation in other respiratory conditions e.g. life-threatening asthma / pulmonary oedema
- sedative drugs: benzodiazepines, opiate overdose

Respiratory alkalosis

Common causes

- anxiety leading to hyperventilation
- pulmonary embolism
- salicylate poisoning*
- CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- altitude
- pregnancy

*salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis



Lung fibrosis

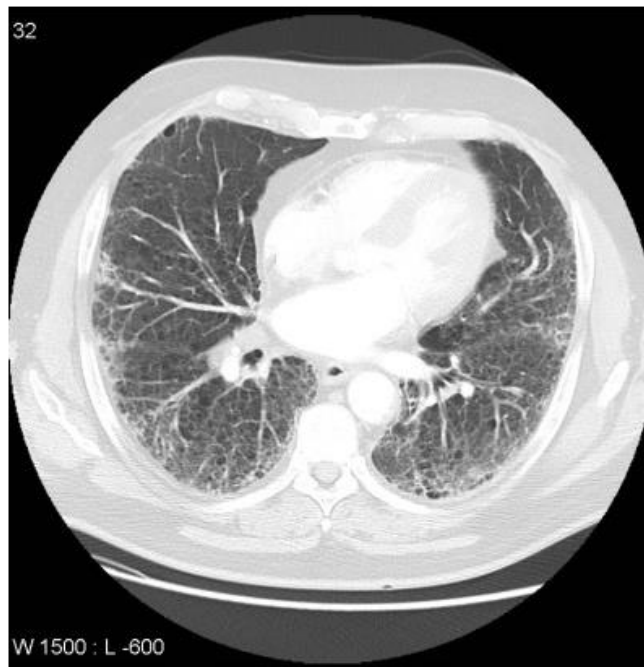
It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis

Fibrosis predominately affecting the lower zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis)
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis



© Image used on license from Radiopaedia

CT scan showing advanced pulmonary fibrosis including 'honeycombing'



Silicosis

Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica). It is a risk factor for developing TB (silica is toxic to macrophages).

Occupations at risk of silicosis

- mining
- slate works
- foundries
- potteries

Features

- fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes



© Image used on license from Radiopaedia

Chest x-ray from a patient with silicosis. Note the bilateral diffuse upper lobe reticular shadowing superimposed with occasional scattered mass

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins
- farmers lung: spores of *Saccharopolyspora rectivirgula* (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute: occur 4-8 hrs after exposure, SOB, dry cough, fever
- chronic

Investigation

- chest x-ray: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- blood: NO eosinophilia

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*



Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome



Sarcoidosis: management

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent.

Indications for steroids

- patients with chest x-ray stage 2 or 3 disease who have moderate to severe or progressive symptoms. Patients with asymptomatic and stable stage 2 or 3 disease who have only mildly abnormal lung function do not require treatment
- hypercalcaemia
- eye, heart or neuro involvement

Bilateral hilar lymphadenopathy

The most common causes of bilateral hilar lymphadenopathy are sarcoidosis and tuberculosis

Other causes include:

- lymphoma/other malignancy
- pneumoconiosis e.g. berylliosis
- fungi e.g. histoplasmosis, coccidioidomycosis

Next question >

Lofgren's syndrome

Lofgren's syndrome is an acute form sarcoidosis characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia.

It typically occurs in young females and carries an excellent prognosis.

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset, symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- CXR: stage III-IV features
- black people

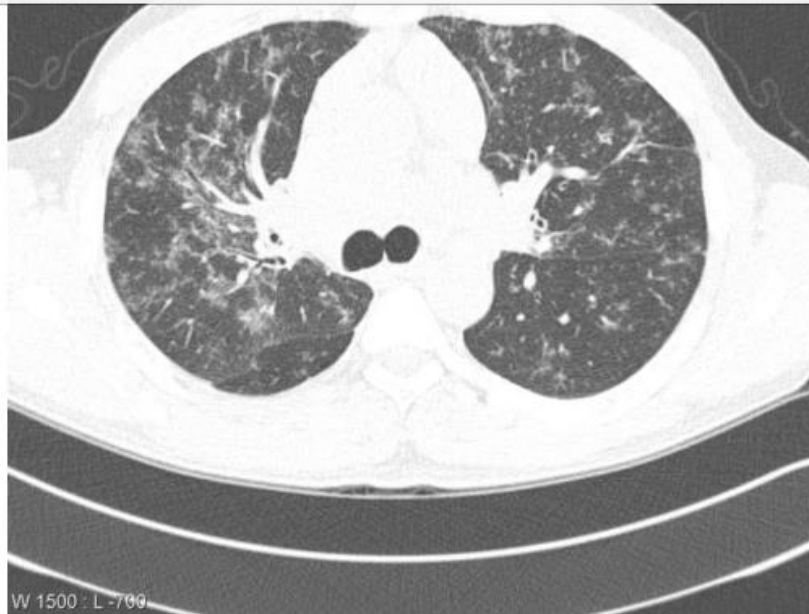
Sarcoidosis: investigation

There is no one diagnostic test for sarcoidosis and hence diagnosis is still largely clinical. ACE levels have a sensitivity of 60% and specificity of 70% and are therefore not reliable in the diagnosis of sarcoidosis although they may have a role in monitoring disease activity. Routine bloods may show hypercalcaemia (seen in 10% of patients) and a raised ESR

A chest x-ray may show the following changes:

- stage 0 = normal
- stage 1 = bilateral hilar lymphadenopathy (BHL)
- stage 2 = BHL + interstitial infiltrates
- stage 3 = diffuse interstitial infiltrates only
- stage 4 = diffuse fibrosis





© Image used on license from Radiopaedia

Chest x-ray and CT scan showing **stage 2** sarcoidosis with both bilateral hilar lymphadenopathy + interstitial infiltrates. The reticulonodular opacities are particularly noted in the upper zones. Remember that pulmonary fibrosis (which this case has not yet progressed to) may be divided into conditions which predominately affect the upper zones and those which predominately affect the lower zones - sarcoidosis is one of the former. The CT of the chest demonstrates diffuse areas of nodularity predominantly in a peribronchial distribution with patchy areas of consolidation particularly in the upper lobes. There is some surrounding ground glass opacities. No gross reticular changes to suggest fibrosis.

Other investigations*

- spirometry: may show a restrictive defect
- tissue biopsy: non-caseating granulomas
- gallium-67 scan - not used routinely

*the Kveim test (where part of the spleen from a patient with known sarcoidosis is injected under the skin) is no longer performed due to concerns about cross-infection

Questions

What affects

Raised

Lower

57 facts

Score for

8 in a row

Key facts

An 80-year-old

x-ray shows

Granuloma

Pneumonia

A 30-year-old

examined

Asbestos and the lung



Asbestos can cause a variety of lung disease from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They are the most common form of asbestos related lung disease and generally occur after a latent period of 20-40 years.

Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis. As with other forms of lung fibrosis the most common symptoms are shortness-of-breath and reduced exercise tolerance.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Asbestos exposure is a risk factor for lung cancer and also has a synergistic effect with cigarette smoke.



Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF, previously termed cryptogenic fibrosing alveolitis) is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs. Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when no underlying cause exists.

IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal crackles on auscultation
- dry cough
- clubbing

Diagnosis

- spirometry: classically a restrictive picture (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- impaired gas exchange: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a chest x-ray but high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF
- ANA positive in 30%, rheumatoid factor positive in 10% but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. Titres are usually low

Management

- pulmonary rehabilitation
- very few medications have been shown to give any benefit in IPF. There is some evidence that pirfenidone (an antifibrotic agent) may be useful in selected patients (see NICE guidelines)
- many patients will require supplementary oxygen and eventually a lung transplant

Prognosis

- poor, average life expectancy is around 3-4 years



COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild**
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

**symptoms should be present to diagnose COPD in these patients

COPD: causes

Smoking!

Alpha-1 antitrypsin deficiency

Other causes

- cadmium (used in smelting)
- coal
- cotton
- cement
- grain

COPD: management of acute exacerbations

The most common bacterial organisms that cause infective exacerbations of COPD are:

- *Haemophilus influenzae* (most common cause)
- *Streptococcus pneumoniae*
- *Moraxella catarrhalis*

Respiratory viruses account for around 30% of exacerbations, with the human rhinovirus being the most important pathogen.

NICE guidelines from 2010 recommend the following:

- increase frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 7-14 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'



COPD: stable management

NICE updated it's guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2010.

General management

- smoking cessation advice
- annual influenza vaccination
- one-off pneumococcal vaccination

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by the FEV1

FEV1 > 50%

- long-acting beta2-agonist (LABA), for example salmeterol, or:
- long-acting muscarinic antagonist (LAMA), for example tiotropium

FEV1 < 50%

- LABA + inhaled corticosteroid (ICS) in a combination inhaler, or:
- LAMA

For patients with persistent exacerbations or breathlessness

- if taking a LABA then switch to a LABA + ICS combination inhaler
- otherwise give a LAMA and a LABA + ICS combination inhaler

Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot use inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Mucolytics

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question >

Next question >

COPD: long-term oxygen therapy

The 2010 NICE guidelines on COPD clearly define which patients should be assessed for and offered long-term oxygen therapy (LTOT). Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day. Oxygen concentrators are used to provide a fixed supply for LTOT.

Assess patients if any of the following:

- very severe airflow obstruction ($FEV_1 < 30\%$ predicted). Assessment should be 'considered' for patients with severe airflow obstruction (FEV_1 30-49% predicted)
- cyanosis
- polycythaemia
- peripheral oedema
- raised jugular venous pressure
- oxygen saturations less than or equal to 92% on room air

Assessment is done by measuring arterial blood gases on 2 occasions at least 3 weeks apart in patients with stable COPD on optimal management.

Offer LTOT to patients with a pO_2 of < 7.3 kPa or to those with a pO_2 of 7.3 - 8 kPa and one of the following:

- secondary polycythaemia
- nocturnal hypoxaemia
- peripheral oedema
- pulmonary hypertension

Next question >

B *I*

A ▾ ▮ ▮ ▮ ▮

T! ▾ ▮ ▮

Next question >

Oxygen therapy

The British Thoracic Society updated its guidelines on emergency oxygen therapy in 2017. The following selected points are taken from the guidelines. Please see the link provided for the full guideline.

In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills. The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets

- acutely ill patients: 94-98%
- patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients

- prior to availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO₂ is normal

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- myocardial infarction and acute coronary syndromes
- stroke
- obstetric emergencies
- anxiety-related hyperventilation

Next question >

B *I*

A ▾ ▾ ▾ ▾ ▾

T ▾ ▾ ▾

Klebsiella

Klebsiella pneumoniae is a Gram-negative rod that is part of the normal gut flora. It can cause a number of infections in humans including pneumonia (typically following aspiration) and urinary tract infections.

Features of *Klebsiella pneumoniae*

- more common in alcoholic and diabetics
- may occur following aspiration
- 'red-currant jelly' sputum
- often affects upper lobes

Prognosis

- commonly causes lung abscess formation and empyema
- mortality is 30-50%



A human neutrophil interacting with *Klebsiella pneumoniae* (pink), a multidrug-resistant bacterium that causes severe hospital infections.
Credit: NIAID



Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus* spores. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

- bronchoconstriction: wheeze, cough, dyspnoea
- bronchiectasis (proximal)

Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to *Aspergillus*
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE



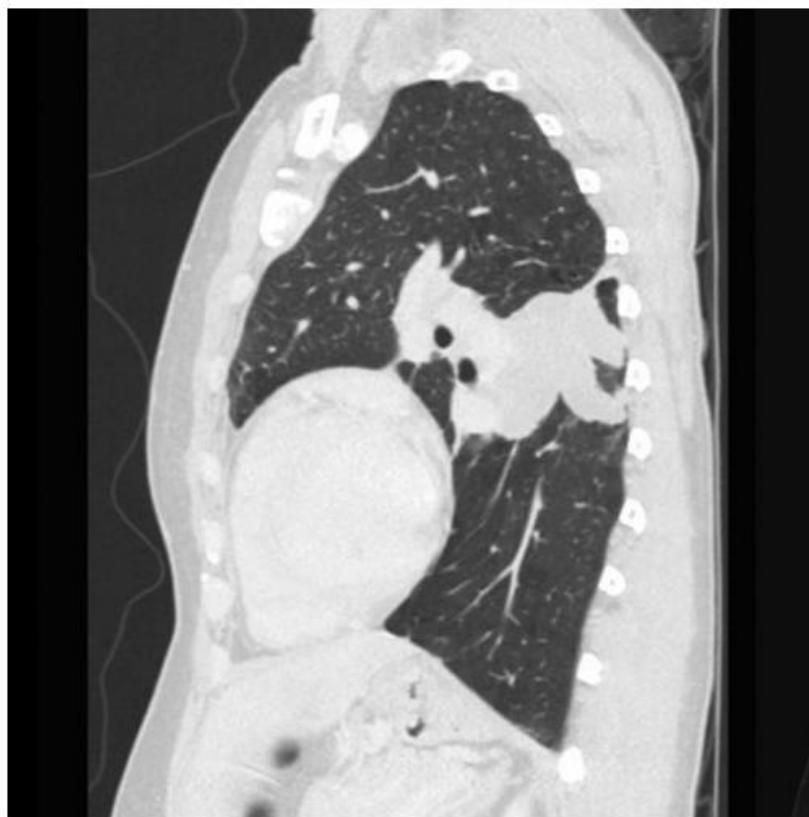
© Image used on license from Radiopaedia

Chest x-ray of a 40-year-old woman with ABPA demonstrating a mass overlying the left hilum. In the right upper parahilar region a few ring shadow / tram track opacities are also noted, suggestive of



© Image used on license from Radiopaedia

Chest x-ray of a 40-year-old woman with ABPA demonstrating a mass overlying the left hilum. In the right upper parahilar region a few ring shadow / tram track opacities are also noted, suggestive of bronchiectasis



© Image used on license from Radiopaedia

CT scan from the same patient. CT reveals a branching lesion in the superior segment of the left lower lobe with classic finger in glove appearance which represents of mucous filling dilated bronchi (i.e. bronchocoeles). Bronchocoeles are a common feature of allergic bronchopulmonary aspergillosis (ABPA)

Management

- steroids
- itraconazole is sometimes introduced as a second line agent

Kartagener's syndrome

Acanth

Polycyt

ACTH s

Hypone

Hypert

Lambe

Kartagener's syndrome (also known as primary ciliary dyskinesia) was first described in 1933 and most frequently occurs in examinations due to its association with dextrocardia (e.g. 'quiet heart sounds', 'small volume complexes in lateral leads')

Pathogenesis

- dynein arm defect results in immotile cilia

Features

- dextrocardia or complete situs inversus
- bronchiectasis
- recurrent sinusitis
- subfertility (secondary to diminished sperm motility and defective ciliary action in the fallopian tubes)

Drugs used in respiratory medicine

Drug	Mechanism of action	Notes
Salbutamol	Beta receptor agonist	• Short-acting inhaled bronchodilator. Relaxes bronchial smooth muscle through effects on beta 2 receptors • Used in asthma and chronic obstructive pulmonary disease (COPD). • Salmeterol has similar effects but is long-acting
Corticosteroids	Anti-inflammatory	• Inhaled corticosteroids are used as maintenance therapy • Oral or intravenous corticosteroids are used following an acute exacerbation of asthma or COPD
Ipratropium	Blocks the muscarinic acetylcholine receptors	• Short-acting inhaled bronchodilator. Relaxes bronchial smooth muscle • Used primarily in COPD • Tiotropium has similar effects but is long-acting
Methylxanthines (e.g. theophylline)	Non-specific inhibitor of phosphodiesterase resulting in an increase in cAMP	• Given orally or intravenously • Has a narrow therapeutic index
Monteleukast, zafirlukast	Blocks leukotriene receptors	• Usually taken orally • Useful in aspirin-induced asthma

Altitude related disorders

Which one

Rheum

Methot

Ankylo

Bleomy

Asbest

Amioda

57 fact

Score f

12 in a

Key fac

Acetaz

Anxiety

Salbuta

There are three main types of altitude related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary edema (HAPE) or high altitude cerebral edema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache
- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE presents with classical pulmonary oedema features
- HACE presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2010. A pneumothorax is termed primary if there is no underlying lung disease and secondary if there is.

Primary pneumothorax

Recommendations include:

- if the rim of air is < 2cm and the patient is not short of breath then discharge should be considered
- otherwise aspiration should be attempted
- if this fails (defined as > 2 cm or still short of breath) then a chest drain should be inserted
- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Secondary pneumothorax

Recommendations include:

- if the patient is > 50 years old and the rim of air is > 2cm and/or the patient is short of breath then a chest drain should be inserted.
- otherwise aspiration should be attempted if the rim of air is between 1-2cm. If aspiration fails (i.e. pneumothorax is still greater than 1cm) a chest drain should be inserted. All patients should be admitted for at least 24 hours
- if the pneumothorax is less than 1cm then the BTS guidelines suggest giving oxygen and admitting for 24 hours
- regarding scuba diving, the BTS guidelines state: *'Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.'*

Iatrogenic pneumothorax

Recommendations include:

- less likelihood of recurrence than spontaneous pneumothorax
 - majority will resolve with observation, if treatment is required then aspiration should be used
 - ventilated patients need chest drains, as may some patients with COPD
-

Pneumonia: assessment and management

Primary care setting

NICE recommends that patients should initially be assessed in primary care using the CRB65 criteria:

Criterion	Marker
C	Confusion (abbreviated mental test score $\leq 8/10$)
R	Respiration rate $\geq 30/\text{min}$
B	Blood pressure: systolic ≤ 90 mmHg and/or diastolic ≤ 60 mmHg
65	Aged ≥ 65 years

Patients are stratified for risk of death as follows:

- 0: low risk (less than 1% mortality risk)
- 1 or 2: intermediate risk (1-10% mortality risk)
- 3 or 4: high risk (more than 10% mortality risk).

NICE recommend, in conjunction with clinical judgement:

- home-based care for patients with a CRB65 score of 0
- hospital assessment for all other patients, particularly those with a CRB65 score of 2 or more.

NICE also mention point-of-care CRP test. This is currently not widely available but they make the following recommendation with reference to the use of antibiotic therapy:

- CRP < 20 mg/L - do not routinely offer antibiotic therapy
- CRP 20 - 100 mg/L - consider a delayed antibiotic prescription
- CRP > 100 mg/L - offer antibiotic therapy

Secondary care setting

Note that in hospital, once blood tests are available the CURB65, rather than the CRB65, can be used. This adds an extra criterion of urea > 7 mmol/L:

Criterion	Marker
C	Confusion (abbreviated mental test score $\leq$ 8/10)
U	urea > 7 mmol/L
R	Respiration rate $\geq$ 30/min
B	Blood pressure: systolic $\leq$ 90 mmHg and/or diastolic $\leq$ 60 mmHg
65	Aged $\geq$ 65 years

NICE recommend, in conjunction with clinical judgement:

- consider home-based care for patients with a CURB65 score of 0 or 1 - low risk (less than 3% mortality risk)
- consider hospital-based care for patients with a CURB65 score of 2 or more - intermediate risk (3-15% mortality risk)
- consider intensive care assessment for patients with a CURB65 score of 3 or more - high risk (more than 15% mortality risk)

Investigations

- chest x-ray
- in intermediate or high-risk patients NICE recommend blood and sputum cultures, pneumococcal and legionella urinary antigen tests
- CRP monitoring is recommend for admitted patients to help determine response to treatment

Management of low-severity community acquired pneumonia

- amoxicillin is first-line

Management of low-severity community acquired pneumonia

- amoxicillin is first-line
- if penicillin allergic then use a macrolide or tetracycline
- NICE now recommend a 5 day course of antibiotics for patients with low severity community acquired pneumonia

Management of moderate and high-severity community acquired pneumonia

- dual antibiotic therapy is recommended with amoxicillin and a macrolide
- a 7-10 day course is recommended
- NICE recommend considering a beta-lactamase stable penicillin such as co-amoxiclav, ceftriaxone or piperacillin with tazobactam and a macrolide in high-severity community acquired pneumonia

Discharge criteria and advice post-discharge

NICE recommend that patients are not routinely discharged if in the past 24 hours they have had 2 or more of the following findings:

- temperature higher than 37.5°C
- respiratory rate 24 breaths per minute or more
- heart rate over 100 beats per minute
- systolic blood pressure 90 mmHg or less
- oxygen saturation under 90% on room air
- abnormal mental status
- inability to eat without assistance.

They also recommend delaying discharge if the temperature is higher than 37.5°C.

NICE recommend that the following information is given to patients with pneumonia in terms of how quickly their symptoms should resolve:

Time	Progress
1 week	Fever should have resolved
4 weeks	Chest pain and sputum production should have substantially reduced
6 weeks	Cough and breathlessness should have substantially reduced
3 months	Most symptoms should have resolved but fatigue may still be present
6 months	Most people will feel back to normal.





Next question >

Pleural effusion: causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia (liver disease, nephrotic syndrome, malabsorption)
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection: pneumonia (most common exudate cause), TB, subphrenic abscess
- connective tissue disease: RA, SLE
- neoplasia: lung cancer, mesothelioma, metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

Next question >

B I

Pleural effusion: investigation and management

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion.

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- contrast CT is now increasingly performed to investigate the underlying cause, particularly for exudative effusions

Pleural aspiration

- as above, ultrasound is recommended to reduce the complication rate
- a 21G needle and 50ml syringe should be used
- fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology

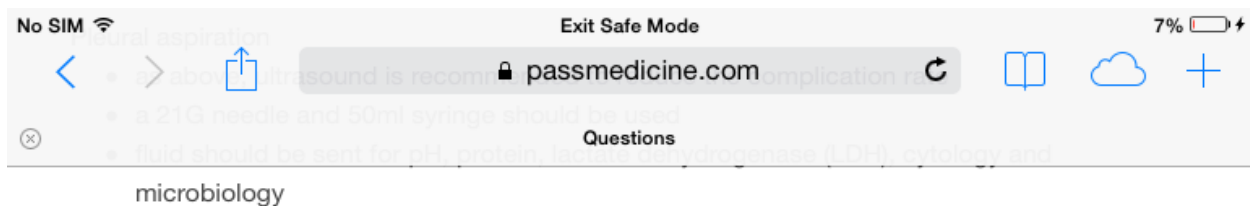
Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. The BTS recommend using the criteria for borderline cases:

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
- pleural fluid protein divided by serum protein >0.5
- pleural fluid LDH divided by serum LDH >0.6
- pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation
- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

Pleural infection



Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. The BTS recommend using the criteria for borderline cases:

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
- pleural fluid protein divided by serum protein >0.5
- pleural fluid LDH divided by serum LDH >0.6
- pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation
- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

Pleural infection

All patients with a pleural effusion in association with sepsis or a pneumonic illness require diagnostic pleural fluid sampling

- if the fluid is purulent or turbid/cloudy a chest tube should be placed to allow drainage
- if the fluid is clear but the **pH is less than 7.2 in patients with suspected pleural infection a chest tube should be placed**

Management of recurrent pleural effusion

Options for managing patients with recurrent pleural effusions include:

- recurrent aspiration
- pleurodesis
- indwelling pleural catheter
- drug management to alleviate symptoms e.g. opioids to relieve dyspnoea

Next question >

Next question >

Bronchiectasis: causes

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. There are a wide variety of causes are listed below:

Causes

- post-infective: tuberculosis, measles, pertussis, pneumonia
- cystic fibrosis
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: selective IgA, hypogammaglobulinaemia
- allergic bronchopulmonary aspergillosis (ABPA)
- ciliary dysketic syndromes: Kartagener's syndrome, Young's syndrome
- yellow nail syndrome



Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- *Klebsiella* spp.
- *Streptococcus pneumoniae*

B I

Rheumatoid arthritis: respiratory manifestations

A variety of respiratory problems may be seen in patients with rheumatoid arthritis:

- pulmonary fibrosis
- pleural effusion
- pulmonary nodules
- bronchiolitis obliterans
- complications of drug therapy e.g. methotrexate pneumonitis
- pleurisy
- Caplan's syndrome - massive fibrotic nodules with occupational coal dust exposure
- infection (possibly atypical) secondary to immunosuppression

Chest x-ray: cavitating lung lesion

Differential

- abscess (Staph aureus, Klebsiella and *Pseudomonas*)
- squamous cell lung cancer
- tuberculosis
- Wegener's granulomatosis
- pulmonary embolism
- rheumatoid arthritis
- aspergillosis, histoplasmosis, coccidioidomycosis

Cryptogenic organizing pneumonia

Cryptogenic organizing pneumonia (COP) is a diffuse interstitial lung disease that affects the distal bronchioles, respiratory bronchioles, alveolar ducts and alveolar walls. It affects 6-7 people per 100,000. The aetiology is unknown.

Males and females are equally affected and tend to present in the 5th or 6th decade of life and is not associated with smoking. Patients typically present with a cough, shortness of breath, fever and malaise. Symptoms can be present for weeks or months. There is often a history of non-response to antibiotics. Haemoptysis is rare. Clinical examination is often normal but inspiratory crackles can be heard. Wheeze and clubbing are rare.

Bloods show a leukocytosis and an elevated ESR and CRP. Imaging typically shows bilateral patchy or diffuse consolidative or ground glass opacities. Lung function tests are most commonly restrictive but can be obstructive or normal. The transfer factor is reduced.

Diagnosis is clinical. Treatment is watch and wait if mild or high dose oral steroids if severe.

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

Consequence

- daytime somnolence
- hypertension

SIGN guidelines for the diagnosis and management of patients with OSAHS were published in 2003

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

- sleep studies - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- CPAP is first line for moderate or severe OSAHS
- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- limited evidence to support use of pharmacological agents

Next question >